the patient, will take him by force if necessary to the psychopathic ward.

The patient is held in this ward a varying period of days to permit examination by licensed medical examiners recognized by the state as especially equipped to render such services. After a trial or hearing before a Superior Court Judge, the patient may be discharged as not mentally ill, committed to a state hospital, or paroled under the psychopathic parole department. Should the patient or his attorney demand a jury trial within five days of the hearing, this will be given him. While awaiting such a trial, the patient is usually held in the psychopathic ward. He may, however, be released to his family or to a private sanitarium.

The result of such a jury trial may be accepted as final, or in the event that the authorities believe a dangerous person has been freed by a jury of his peers, they may arrange another trial by swearing out another petition. The procedures, first outlined, always involve the presence of the patient in the psychopathic ward at least for examination, and his appearance before the judge.

About two years ago, a change was made in the law and another procedure is coming into more general use. A patient may be examined by two especially licensed medical examiners who are either members of the old State Lunacy Commission, or newer appointees of the Judge presiding over the psychopathic court, and these physicians then may fill out the Petition to Detain. This paper is then sent to the commission. A psychopathic parole officer visits the patient, and if his or her investigation is satisfactory, approves the Petition to Detain, which then goes to the judge. The judge may parole to a private sanitarium, or commit to a state institution without the patient going to the psychopathic ward or appearing in court. This procedure is greatly appreciated by many families who, without great justification, dread the ordeal of the psychopathic ward for the patient. It is well for the internist to know that such a procedure is possible.

Voluntary commitment is permissible, but is discouraged by state institutional authorities because such commitments rarely stand up for a long enough period to be really helpful to the patient. Furthermore, it is to be noted that the law states that a person signing a voluntary commitment must be mentally competent. This, as I am sure you can realize, leaves a loophole for controversy in court if the state hospital authorities do not agree with the patient as to the wisdom or propriety of his discharge from their institution.

Families or friends of patient's frequently ask the physician, when he has outlined the above procedures, if such steps are really necessary. They suggest the patient be given sedative medication or otherwise restrained, and taken when unconscious or in restraint to a private sanitarium. Is it ever justifiable to thus shanghai a patient without evoking legal aid? The answer is, no. Such a procedure is never justifiable and actually never without danger to the physician advising or taking part in such a course of action. It has been accepted in a Superior Court of California recently, that if any form of restraint is applied to any patient for any appreciable period of time—even though the restraint be removed instantly the patient demands it—the person applying the restraint may be sued for damages, as may the institution where the restraint was applied. This is, of course, provided the full legal requirements have not been previously fulfilled.

My advice to you all is, never take such responsibility. After all, we physicians did not make the laws nor were we consulted when they were being made. We are, however, expected to obey them. Medicinal or physical restraint may only be safely used after observance of the legal formalities.

The physician may be asked to sign the Petition to Detain. My advice is that he refuse to do so. Though the law now states that he cannot be sued for so doing, there is at the present time such a suit in the courts. The signing of the Petition to Detain is the direct responsibility of the next of kin or of the most interested party. If no such person is available, and the situation is urgent, call the police and place the responsibility on their shoulders. If the case is not an emergency one, the Los Angeles Police Force has an officer to whom these problems may be referred. This officer in charge of psychiatric matters for the police will, if notified, investigate the situation and take the action he deems necessary. The physician has done his duty, and is relieved of further responsibility in the matter.

In summary, the relationship between the internist and the psychiatrist is rightfully a close one. Still greater coöperation and mutual understanding are desirable. Emergency cases falling to the internist should be handled with due emphasis placed on the legal requirements just discussed.

1136 W. Sixth Street.

ERYTHROBLASTOSIS FETALIS*

REPORT OF CASE

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INTRODUCTION.—Between one in 500 and one in 1000[1,2] newborn babies show a marked dysfunction of the blood-forming and blood-destroying systems of the body, which may be exhibited in a number of ways, all grouped under the general heading of erythroblastosis fetalis. The common feature of these cases is the presence of (1) abnormal islands of blood-forming tissue in the liver, spleen, kidneys, and others organs;

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(2) excessive numbers of immature erythrocytes in the circulating blood; (3) increased destruction of erythrocytes; and (4) a distinct tendency for the disease to appear in successive children of a family.

The three forms which the disease may exhibit are (1) icterus gravis neonatorum; (2) congenital anemia of the newborn; and (3) congenital hydrops, or universal edema of the fetus. Although these three conditions have been known individually for years, it has been only within the last decade that their common background has been recognized. Diamond and his associates,[5] in 1932, reviewed the literature and presented twenty cases to illustrate the point. Despite the underlying pathological process (disturbance of the blood-forming system) and the appearance of anemia in all three conditions, each presents a quite distinct symptomatology.

In icterus gravis the infant may appear entirely normal at birth, but within a few hours or days develops a marked jaundice and anemia, with many circulating nucleated red-blood cells and an enlarged liver and spleen. The bleeding time becomes prolonged, the anemia becomes progressively more severe, and, if untreated, the infant usually dies within one or two weeks.

In congenital anemia, the most striking feature is an anemia of increasing severity, accompanied by marked evidence of blood formation and destruction. The chief difference between this form of erythroblastosis fetalis and icterus gravis is that, in congenital anemia, the anemic process tends to overshadow the jaundice. In this connection Hellman and Hertig,[4] after reviewing 35 cases of erythroblastosis fetalis in 30,000 births, came to the conclusion that congenital anemia is merely a sequel to icterus gravis.

The gravest form of erythroblastosis fetalis is the so-called congenital hydrops, or universal edema of the newborn. The nature of this condition is illustrated by the following case, which was referred by Dr. Edward Liston of Palo Alto.

REPORT OF CASE

Baby girl, W., was born spontaneously at term on August 1, 1940. The prenatal course had been normal, and tests for syphilis and Bacillus abortus agglutinins on the mother had been negative. The mother, aged 29, had had three previous pregnancies. The first child, born in 1933, appeared normal at birth and had a negative cord Wassermann, but died a few days later. A five-and-one-half month abortion occurred in 1934 and a three month abortion in 1936.

Immediately upon delivery of Baby W., it was obvious that the child was not normal, and consultation was sought. The infant was examined by one of us (R.D.C.) fifteen minutes after birth. It was extremely pale, with large ecchymoses over the face and smaller ecchymotic spots on the trunk and extremities. The body was mildly edematous and the placenta markedly so, being about twice normal size. Respirations were irregular. The liver edge was smooth and firm, and extended below the umbilicus. A diagnosis of erythroblastosis fetalis, hydrops type, was made, blood specimens were taken for examination, and the infant was immediately given an intravenous transfusion of sixty cc. of matched uncitrated blood. Despite this treatment, death occurred within one hour after birth.

The laboratory reports showed a marked anemia with a red count of 900,000 and hemoglobin of 32 per cent (5.5 grams). There was a considerable excess of nucleated red cells, and there were many abnormally large red cells. Very few platelets were noted. The leucocyte count was 26,000, of which 12 per cent were immature forms. The coagulation time was seven minutes, and the bleeding time over five and one-half minutes. The blood was type II (Moss). An autopsy and pathological examination were done by one of us (B.L.D.), with the following findings: An unusually large placenta, weighing 1110 grams, was composed of huge firm cotyledons, white on section, which were separated by deep fissures. The broad cord and an accessory placenta added another 190 grams. Histologically the villi were large and had highly cellular edematous vacuolate stroma. Some of the peripherally arranged blood vessels contained nucleated red cells. In general, the microscopic appearance was more that of a midterm than a full-term placenta, and was typical of congenital hydrops. The stroma of the cord was edematous, and nucleated red cells were seen in the vein, but the arteries were empty.

The markedly livid newborn female body was slightly edematous and, with the exception of a greatly distended abdomen, showed no superficial congenital malformation. It weighed 2604 grams. The fetalplacental ratio thus was 2:1 instead of the usual 6:1. Microscopically, the myocardium was composed of the usual immature cardiac muscle cells, with scattered small interacellular glycogen deposits revealed by special stain. The amount was not sufficient to indicate glycogen storage disease. A few normoblasts were seen in the capillaries, but no blood islands were demonstrated.

A tremendously enlarged liver weighed 380 grams and distended the abdomen. Its capsule was smooth and glistening. The parenchyma was a dark purple. Many large clumps of nucleated red cells were seen in the sections. These filled and distended the sinuses, which were surrounded by parenchymal cells laden with granular brown pigment, indicating blood destruction.

The large eighty gram spleen was tense with blood. Myriads of immature erythrocytes in a fine collagenous stroma were seen in the sections.

Kidneys of normal size, weighing twenty grams each, showed clumps of nucleated red blood cells.

Elsewhere clumps of young red cells were seen in the lymph nodes and adrenals, and sections of the bone marrow showed very active blood formation.

To summarize: the pathological examination revealed edema of the placenta and marked evidence of blood destruction and widely-scattered blood formation.

COMMENT

Etiology.—It is obvious from the above descriptions that there is a common underlying pathology of the three forms of erythroblastosis fetalis, namely abnormal blood formation and destruction. As to the actual etiology of the disease, numerous theories have been advanced. One of the earliest (1935)[3] was the persistence of embryonal blood-forming foci in various organs. Other theories have included the exhaustion of a maternal hormone necessary for the stimulation of fetal blood formation.[6] A dominant mutation has also been suggested as the cause of the disease,[7] but the statistics on which this theory is based have been challenged.[8]

Diamond and his associates[5] have explained a number of the symptoms of the disease, and have pointed out that the icterus is due to abnormal destruction of immature red cells, plus clogging of the liver with blood pigment to produce an

obstructive jaundice. They have suggested that the edema is due to capillary damage produced by anemia and anoxemia. Weinberg [9] found elevated blood ureas in three cases of erythroblastosis fetalis, and suggested renal failure as the cause of the edema.

An interesting explanation of the etiology of the disease was made in 1938 by Darrow,[8] who argued that the fundamental pathological processes of erythroblastosis fetalis are abnormal destruction of erythrocytes and dysfunction of the liver due to injury. She attributed both the red cell destruction and liver damage to anaphylaxis, that is, sensitization of the fetus to antibodies formed in the mother's blood, following escape of fetal hemoglobin into the maternal circulation. This concept is especially interesting in the light of work reported this year by Levine and his associates,[19] on transfusion accidents in recently-delivered mothers due to atypical blood agglutinins. In studying five such cases, they found that three had given birth to infants suffering from erythroblastosis fetalis. Their hypothesis is that the mother becomes immunized to certain fetal factors possibly inherited from the father, and that, under certain conditions, the resulting agglutinins are able to pass the placental barrier and enter the fetal circulation, where they act upon "the blood cells and, perhaps, tissue cells of the fetus."

Diagnosis.—In severe cases diagnosis is often possible at birth, especially in congenital hydrops, where the appearance is startling, with icterus or marked pallor, edema, hemorrhagic spots over the body, and a huge placenta. A yellow amniotic fluid and vernix caseosa have been referred to as diagnostis guides, but they are apparently unreliable.[4] As a matter of fact, it has been stated that congenital hydrops can be diagnosed prenatally by use of the x-ray. In general, erythroblastosis fetalis should be suspected if the placenta is unusually large, or if it is pale and friable [17, 18], as in our patient.

Cases of icterus gravis and congenital anemia are sometimes impossible to diagnose at birth,[18] but soon develop a jaundice and anemia far more severe than are found in ordinary icterus neonatorum, together with an enlarged liver and spleen. The only certain methods of early diagnosis in these two forms of erythroblastosis require skilled pathological or laboratory study. The first is a microscopical examination of the placenta, which reveals characteristic enlargement of the placental villi, and epithelial vacuolization.[11] The second is counting the nucleated red cells, which has been found significant by Monfort and Brancato,[14] who noted that an excess of nucleated erythrocytes at birth, with failure to drop to normal by the second day, was diagnostic of erythroblastosis fetalis.

Therapy.—Once the diagnosis has been established, treatment becomes a matter of urgency, at least in cases of icterus gravis and congenital anemia. So far as we know, no therapeutic measures have saved any case of congenital hydrops.[4] The only procedure of proven efficacy in icterus gravis and congenital anemia is the use of matched blood transfusions. These should be intravenous rather than intramuscular, of sixty to eighty cc. in volume, started early, and repeated frequently —every day or so. Until more is known concerning the rôle of isoimmunization in the etiology of erythroblastosis fetalis, the donor probably should not be a member of the immediate family. Transfusions, if begun early, may prevent the anemia of icterus gravis, and may abort the abnormal erythroblastosis, according to Cohen.[15] Hellman and Hertig [4] reported that, in their series of twenty cases of icterus gravis, the deaths of ten were due to failure to transfuse, or to transfusions given inadequately or too late.

Recently Mayman [16] has reported on the use of vitamin K in the treatment of one case of icterus gravis. The vitamin was given on the ninth postnatal day, and within twelve hours the stools, which had been clay-colored, became and remained yellow. After several more days the jaundice began to clear and the baby began to improve generally. Whether this clearing up of what was apparently in part an obstructive jaundice was due to the vitamin K or would have occurred anyway, remains a matter for conjecture until further cases are reported. Personally, we feel that transfusions should be relied upon.

Prophylaxis.—As to prophylaxis, very little can be suggested. Adams and Cochrane [17] reported a family of three children, all born to the same mother. The first died two hours after birth of cerebral hemorrhage. The second developed icterus gravis, which was successfully treated with frequent blood transfusions and concentrated liver extract intramuscularly. During the last seven months of her third pregnancy, the mother received repeated intramuscular injections of concentrated liver extract and gave birth to a normal infant. Since the incidence of recurrences of icterus gravis in families is about eighty per cent, this use of liver extract prophylactically may have had some significance. At any rate, the only other certain method of avoiding subsequent cases of erythroblastosis fetalis in a family is to prevent further pregnancies.

In Conclusion.—As a final word, we should like to point out, once again, a few of the more important features of the disease. First, it may assume any one of the three forms which we have discussed; second, a study of the literature pretty well indicates that if we lump the three forms together for statistical purposes, erythroblastosis fetalis is not an exceptionally rare disease; third, there is a very distinct familial tendency, especially in icterus gravis; and, finally, the only treatment of proven value is repeated blood transfusions.

SUMMARY

1. Icterus gravis neonatorum, congenital anemia, and congenital hydrops are manifestations of erythroblastosis fetalis, a congenital disease of the newborn characterized by abnormal blood formation and destruction.

2. The gravest form of the disease is congenital hydrops, a case of which is reported.

3. The various theories of etiology, including isoimmunization, are reviewed.

4. Diagnosis is obvious in congenital hydrops; but in icterus gravis and congenital anemia the characteristic jaundice and anemia may be delayed until a few hours or days after birth.

5. The only effective therapy is early and frequent intravenous transfusions of blood, preferably from a donor who is not a member of the immediate family.

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PROPOSED PLAN FOR MOBILE BLEEDING UNITS IN NORTHERN CALIFORNIA*

IRWIN MEMORIAL BLOOD BANK OF THE SAN FRANCISCO COUNTY MEDICAL SOCIETY

2180 Washington Street
San Francisco, California
(Telephone Walnut 5600)

To the Editor:—The American Red Cross originally asked for a quota of 10,000 units of blood from the Northern California Procurement Center. This quota is now a thing of the past—war has been declared—quotas are out, and we will keep on drawing blood until this conflict is over.

In order to augment our San Francisco supply, and to allow all Northern California communities large and small to participate in this fundamental Red Cross program we will send out small mobile bleeding units from the Irwin Memorial Blood Bank in San Francisco. This bank has been designated the official procurement center for Northern California.

Each motorized unit will have an ice box capacity for 80 to 120 units of blood, and adequate storage space for the necessary medical and secretarial supplies. The personnel for each unit will consist of one of our Blood Bank nurses, a volunteer chauffeuse, a technical assistant, and a recording secretary.

Our tentative plan for operation is as follows: San Francisco will be the hub of the wheel; towns distant to San Francisco will be plotted on the spokes radiating to the north, to the south, and to the east. One, or more doctors from those communities closest to the hub will be asked to visit the Irwin Memorial Blood Bank to see the technique we have developed for blood-letting, in order that their method will conform with ours.

On a certain day, chosen at least two weeks in advance so that the local Red Cross or some other responsible agency can sign up the requisite number of donors, the mobile bleeding unit will be sent to an adjacent town, and the doctors of that town who have had a "refresher course" in drawing blood at our Bank will perform the actual bleeding. Our specially trained nurse will not only assist the local doctors, but she will be available to answer pertinent questions as to technique etc. While the mobile bank is in operation at the above town two or more doctors from towns yet distant from San Francisco and the town where the drawing is in progress will be asked to attend in order to watch the proceedings and then to actually bleed a few donors. This controlled progression from the center will carry our uniform technique throughout Northern California. The reasons why such a policy must be carried out are:

1. There are too few doctors available, due to the national emergency, to adequately man the vehicles necessary for this large scale program.

2. Expense would be too great; if doctors *were* available for such medical personnel, they would have to be full time and salaried.

3. Voluntary donors will have more confidence in their local doctors and this fact will augment the response.

Medical men in each community will choose those doctors best fitted for intravenous work. This can be accomplished through the various County Medical Societies. Each society must see to it that a proper equalization of effort is made, as the plan must not bear heavily on the few for its success, but all work must be evenly distributed.

The medical profession of Northern California has been asked to help this new widely disseminated plan for collecting blood for the U. S. Army and Navy and to aid in creating a supply to be used in any national disaster. We can point the way for other states, as our plan is unique in its simplicity. Its full success will depend on three factors:

1. Full support of all doctors in making their community 100 per cent donor conscious.

2. Absolute adherance to the technique which we have evolved for drawing blood by the vacuum "closed" method.

3. Vigilant attention to routine orders by all parties concerned so that perfect coordination between Headquarters and all distant drawing points can be scrupulously maintained.

Please keep in touch with your local Red Cross Chapter. Do not hesitate to write me, but in a short while comprehensive instructions will be sent to the proper local medical authorities. Visit our Blood Bank if you are in San Francisco and see the greatly expanded project—you are always welcome.

(Signed) JOHN R. UPTON, M. D., *Secretary-Treasurer, Irwin Memorial Blood Bank; and Technical Supervisor, Red Cross Procurement Center.*

* For other comment on Irwin Blood Bank, see page 9.